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- => s diagnostic
- L1 1290572 DIAGNOSTIC
- => s l1 and schizophrenia
- L2 12154 L1 AND SCHIZOPHRENIA
- => s 12 and platelet
- L3 136 L2 AND PLATELET
- => s 13 and isolectic point
- L4 0 L3 AND ISOLECTIC POINT
- => s 13 and pI
- L5 1 L3 AND PI
- => d 15 all
- L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS
- AN 1999:390463 CAPLUS
- DN 131:16115
- TI Skin test for schizophrenia
- IN Shinitzky, Meir; Deckmann, Michael
- PA Yeda Research and Development Co. Ltd., Israel
- SO PCT Int. Appl., 21 pp.
 - CODEN: PIXXD2
- DT Patent
- LA English
- IC ICM G01N033-68
- CC 9-16 (Biochemical Methods)
 Section cross-reference(s): 14

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

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PRAI IL 1997-122490
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     WO 1998-I
L592
         19981207
     A diagnostic method for assaying schizophrenia in a
AΒ
     subject is provided wherein a prepn. comprising platelet derived
     proteins or fractions thereof having a pI above about 6.5 is
     injected into a subject and the occurrence of delayed type
     hypersensitivity (DTH) reaction at the site of the injection is detd. A
     pos. DTH reaction indicates that the tested subject has a high likelihood
     of being schizophrenic. The protein prepn. used in the diagnostic
     method is also provided as well as a method for its prepn. and a kit for
     use in the diagnosis of schizophrenia using the above method.
ST
     schizophrenia diagnosis skin test
IT
     Blood
     Diagnosis
     Platelet (blood)
     Schizophrenia
         (skin test for schizophrenia)
IT
     Proteins, general, biological studies
     RL: BSU (Biological study, unclassified); BIOL (Biological study)
         (skin test for schizophrenia)
RE.CNT 2
RE
(1) Burbaea, G; Zk Nevropatol Psikhiatr im S S Korsakova 1986, V86(1), P193
    CAPLUS
(2) Yeda Research and Development Company Ltd; WO 9713152 A 1997 CAPLUS
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L3
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1.4
L5
                1 S L3 AND PI
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     2000:241564 CAPLUS
ΑN
DN
     132:288780
     Methods of identifying inverse agonists of the serotonin 2a receptor,
ΤI
     therapeutic and diagnostic methods, and test kit
IN
     Weiner, David; Brann, Mark R.
     Acadia Pharmaceuticals Inc., USA
PΑ
     PCT Int. Appl., 42 pp.
SO
     CODEN: PIXXD2
DT
     Patent
LΑ
     English
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                       19981007
PRAI US 1998-103317
                       19991006
     US 1999-413626
     WO 1999-US21439 19991007
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RE
(1) Eggerickx, D; BIOCHEMICAL JOURNAL 1995, V309, P837 CAPLUS
(3) Herrick, D; WO 9838217 A 1998 CAPLUS
(4) Inst Of Psychiatry; WO 9617081 A 1996 CAPLUS
(6) Shenker, A; NATURE 1993, V365, P652 CAPLUS
(7) Smith, J; WO 9952927 A 1999 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT
=> d his
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          12154 S L1 AND SCHIZOPHRENIA
L3
             136 S L2 AND PLATELET
L4
               0 S L3 AND ISOLECTIC POINT
L5
               1 S L3 AND PI
              85 DUP REMOVE L3 (51 DUPLICATES REMOVED)
Lб
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ΑN
     2000:227858 CAPLUS
DN
     132:260666
ΤI
     Identifying agents that alter mitochondrial permeability transition pores
     and cell death for diagnostic and therapeutic use
     Dykens, James A.; Miller, Scott W.; Ghosh, Soumitra S.; Davis, Robert E.
IN
     Mitokor, USA
PCT Int. Appl., 88 pp.
PA
SO
     CODEN: PIXXD2
DT
     Patent
LΑ
     English
ΙÇ
     ICM G01N033-50
     ICS G01N033-68; A61K031-00; C07C279-26
     1-1 (Pharmacology)
     Section cross-reference(s): 63
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     WO 2000019200
                      A1 20000406
                                           WO 1999-US22261 19990924
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             MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ,
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                       19980925
PRAI US 1998-161172
     WO 1999-US22261 19990924
AB
     Methods are provided for identifying agents that affect mitochondrial
     functions and cell death. Such agents are useful for treating diseases
     assocd. with mitochondrial dysfunction and in methods of identifying a
     risk or presence of such diseases. In particular, the invention relates
     to the loss of mitochondrial membrane potential (.DELTA..PSI.m) during
     mitochondrial permeability transition (MPT) and further provides a
     measurable rate loss function, changes in which are useful e.g. for
     detecting agents that affect one or more mitochondrial functions, for
     detecting mitochondrial diseases, and for studying mol. components of
     mitochondria that regulate MPT.
ST
     mitochondria permeability transition pore therapeutic identification;
     diagnosis mitochondrial disease permeability transition pore; cell death
     mitochondrial permeability therapeutic identification; membrane potential
     mitochondria diagnostic therapeutic identification
IT
     Transport proteins
```

RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (ADP/ATP carrier; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Cyclophilins RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (D; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Apolipoproteins RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process) (E, genotype; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Nervous system (Huntington's chorea; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Brain, disease (MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes); identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Muscle, disease (MERRF (myoclonic epilepsy assocd. with ragged-red muscle fibers); identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Animal cell line (SH-SY5Y, cybrid cell produced with; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Annexins RL: BPR (Biological process); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses) (V, FITC conjugates; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Anion channel RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (VDAC (voltage-dependent anion-selective channel); identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Neurotransmitters RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study) (amino acid; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Diabetes mellitus (and mitochondrial diabetes and deafness; identification of agents

alter mitochondrial permeability transition pores and cell death for

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

TT

ΙT

ΙT

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IT

TΤ

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ΙT

Gene, animal

that.

diagnostic and therapeutic use)

Page 6

(Biological study); PROC (Process)

(bcl-2, Bcl-2 gene family-encoded polypeptide; identification of agents $\ensuremath{\text{gen}}$

that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Membrane potential

(biol.; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Transport proteins

RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (calcium-transporting, mitochondrial calcium uniporter; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use)

IT Platelet (blood)

(cybrid cell produced with; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use)

IT Animal cell

(cybrid cell; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Muscle, disease

(degeneration; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Mitochondria

(diseases; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Nervous system

(dystonia; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Pathogen

(eukaryotic; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Indicators

(for inner mitochondrial membrane potential; identification of agents that alter mitochondrial permeability transition pores and cell death for **diagnostic** and therapeutic use)

IT Eye, disease

(hereditary optic atrophy; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use)

IT Cell proliferation

(hyperproliferative disease; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use)

IT Affinity labeling

Alzheimer's disease Anti-Alzheimer's agents Antidiabetic agents Antiparkinsonian agents Antipsychotics Antitumor agents Apoptosis

Brain, disease Cell death Diagnosis Drug delivery systems Drug screening Electron transport system, biological Fluorometry Genotypes Insect (Insecta) Ionophores Lepidoptera Mitochondria Necrosis Neoplasm Nucleic acid library Parkinson's disease Plant (Embryophyta) Psoriasis Schizophrenia (identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) TΨ DNA RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) ITCell proliferation (inhibitors; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) IT Mitochondria (inner membrane; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) Membrane, biological (inner mitochondrial; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) ΙT Biological transport (intracellular, phosphatidylserine; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) IT Acidosis (lactic acidosis; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) IT Time-of-flight mass spectrometry (laser-induced photodesorption; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) ΙT Deafness (mitochondrial diabetes and deafness; identification of agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use) ΙT Amino acids, biological studies

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RL: BAC (Biological activity or effector, except adverse); BIOL
     (Biological study)
        (neurotransmitter; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
TΫ
     Parasite
        (of human; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
IΤ
     Eukaryote (Eukaryotae)
        (pathogen; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
ÌТ
     Benzodiazepine receptors
     RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
        (peripheral; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
TТ
     Biological transport
        (permeation; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
ΤТ
     Laser ionization mass spectrometry
        (photodesorption, matrix-assisted; identification of agents that alter
        mitochondrial permeability transition pores and cell death for
      diagnostic and therapeutic use)
ΤТ
     Laser desorption mass spectrometry
        (photoionization, matrix-assisted; identification of agents that alter
        mitochondrial permeability transition pores and cell death for
      diagnostic and therapeutic use)
ΙT
     Laser desorption mass spectrometry
        (time-of-flight; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
TT
     Antibodies
     RL: BPR (Biological process); BUU (Biological use, unclassified); BIOL
     (Biological study); PROC (Process); USES (Uses)
        (to cytochrome c; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
ΙT
     Phosphatidylserines
     RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
        (translocation; identification of agents that alter mitochondrial
        permeability transition pores and cell death for diagnostic
        and therapeutic use)
ΙT
     145037-81-6, Rhod 2
     RL: BPR (Biological process); BUU (Biological use, unclassified); BIOL
     (Biological study); PROC (Process); USES (Uses)
        (Rhod 2; identification of agents that alter mitochondrial
permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
TΤ
     51-83-2, Carbachol
                          56-86-0, L-Glutamic acid, biological studies
     6384-92-5, N-Methyl-D-aspartic acid
                                          11076-19-0, Bongkrekic acid
     11103-72-3, Ruthenium red 17754-44-8, Atractyloside
                                                            56092-81-0,
     Ionomycin 67526-95-8, Thapsigargin 79217-60-0, Cyclosporin
     169332-61-0
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     RL: BAC (Biological activity or effector, except adverse); BIOL
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(Biological study)
        (identification of agents that alter mitochondrial permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
     9007-43-6, Cytochrome c, biological studies
                                                   122191-40-6, Caspase 1
     169592-56-7, Caspase 3 186322-81-6, Caspase
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     process); BIOL (Biological study); PROC (Process)
        (identification of agents that alter mitochondrial permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
IT
     102-02-3, 1-Phenylbiguanide
     RL: BAC (Biological activity or effector, except adverse); THU
     (Therapeutic use); BIOL (Biological study); USES (Uses)
        (identification of agents that alter mitochondrial permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
ΤТ
     7440-70-2, Calcium, biological studies
     RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
        (identification of agents that alter mitochondrial permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
IT
     2156-29-8
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                                  18198-39-5, Tetraphenylphosphonium
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     (Biological study); PROC (Process); USES (Uses)
        (identification of agents that alter mitochondrial permeability
        transition pores and cell death for diagnostic and
        therapeutic use)
ΙT
     9001-15-4, Creatine kinase
     RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
        (mitochondrial intermembrane; identification of agents that alter
        mitochondrial permeability transition pores and cell death for
      diagnostic and therapeutic use)
IT
     9001-51-8, Hexokinase
     RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
        (mitochondrial-assocd.; identification of agents that alter
        mitochondrial permeability transition pores and cell death for
      diagnostic and therapeutic use)
RE.CNT
(1) Beal, M; Biochimica et Biophysica Acta 1998, V1366(1-2), P211 CAPLUS
(2) Diamond, J; GB 1410925 A 1975 CAPLUS
(3) Friberg, H; Journal of Neuroscience 1998, V18(14), P5151 CAPLUS
(4) Hirsch, T; Cell Biology and Toxicology 1998, V4(2), P141
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ΑN
     1999:390463 CAPLUS
DN
     131:16115
TI
    Skin test for schizophrenia
ΙN
    Shinitzky, Meir; Deckmann, Michael
    Yeda Research and Development Co. Ltd., Israel
     PCT Int. Appl., 21 pp.
SO
     CODEN: PIXXD2
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DT
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LΑ
     English
IC
     ICM G01N033-68
     9-16 (Biochemical Methods)
     Section cross-reference(s): 14
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                     KIND DATE
                                          APPLICATION NO. DATE
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     WO 1998-I
L592
        19981207
AB
     A diagnostic method for assaying schizophrenia in a
     subject is provided wherein a prepn. comprising platelet derived
     proteins or fractions thereof having a pI above about 6.5 is
     injected into a subject and the occurrence of delayed type
     hypersensitivity (DTH) reaction at the site of the injection is detd. A
     pos. DTH reaction indicates that the tested subject has a high likelihood
     of being schizophrenic. The protein prepn. used in the
     diagnostic method is also provided as well as a method for its
     prepn. and a kit for use in the diagnosis of schizophrenia using
     the above method.
ST
     schizophrenia diagnosis skin test
ΙT
     Blood
     Diagnosis
     Platelet (blood)
     Schizophrenia
     Skin
        (skin test for schizophrenia)
     Proteins, general, biological studies
     RL: BSU (Biological study, unclassified); BIOL (Biological study)
        (skin test for schizophrenia)
RE.CNT
(1) Burbaea, G; Zk Nevropatol Psikhiatr im S S Korsakova 1986, V86(1), P193
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TΙ
     [Brain isoforms of creatine kinase in health and mental diseases:
    Alzheimer's disease and schizophrenia].
    Mozgovaia izoforma kreatinfosfokinazy v norme i pri psikhicheskikh
    zabolevaniiakh (bolezn' Al'tsgemera, shizofreniia).
```

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ΑIJ
     Burbaeva GSh; Savushkina O K; Dmitriev A D
SO
     VESTNIK ROSSIISKOI AKADEMII MEDITSINSKIKH NAUK, (1999) (1) 20-4. Ref: 39
     Journal code: BL9. ISSN: 0869-6047.
CY
     RUSSIA: Russian Federation
     Journal; Article; (JOURNAL ARTICLE)
חיים
     General Review; (REVIEW)
     (REVIEW, TUTORIAL)
LΑ
     Russian
     199906
EM
EW
     19990601
AB
     The paper analyzes the authors' own findings and the data available in
the
     literature on the intensity, site, and possible causes of impairment of
     the creatine-creatine phosphate system of brain energy metabolism in
     mental diseases, such as Alzheimer's disease (AD) and
     schizophrenia. Examining the level of cytosolic BB creatine kinase
     in postmortem AD and schizophrenic's brain structures showed a
significant
     decrease in BB creatine kinase as compared with the similar control brain
     structures. There was the maximum decline in AD cases. It was
considerable
     as compared with both the control and schizophrenic groups (p < 0.01).
The
     decrement was revealed by various techniques, including the determination
     of activity, immunological responsiveness and the analysis of
     two-dimensional protein maps. Immunocytochemical investigation
     indicated a decrease in responses to BB creatine kinase, mainly in
     astrocytes. The reduction in cytosolic BB creatine kinase levels is not a
     result of age, postmortem delay, or psychotic therapy. The causes of
lower
     BB creatine kinase levels in the cell cytosol of the postmortem brain in
     mental pathology are discussed. The decrement in cytosolic BB creatine
     kinase in AD and schizophrenia occurs not only in the brain, but
     also in the peripheral tissues which contain BB creatine kinase. In all
     cases, it is greater in AD than in schizophrenia. Using
     immunosorbents with monoclonal antibodies to M-creatine kinase and to
     B-creatine kinase subunits makes it possible detect BB-creatine kinase in
     the extracts of human peripheral lymphocytes and platelets. A
     study of whether there is a relationship between the clinical data of
     mental patients and the level of BB creatine kinase in their blood
     elements is assumed to be useful in evaluating BB creatine kinase as a
     prognostic/diagnostic marker of mental diseases.
     Check Tags: Comparative Study; Human
     Alzheimer Disease: DI, diagnosis
     *Alzheimer Disease: EN, enzymology
     Biological Markers
     *Brain: EN, enzymology
     *Creatine Kinase Isoenzymes: ME, metabolism
     Diagnosis, Differential
     English Abstract
     Schizophrenia: DI, diagnosis
     *Schizophrenia: EN, enzymology
CN
    EC 2.7.3.- (Creatine Kinase Isoenzymes); 0 (Biological Markers)
L8
    ANSWER 4 OF 5 CAPLUS COPYRIGHT 2001 ACS
ΑN
     1998:568669
                 CAPLUS
DN
     129:185107
TT
    Cloning and cDNA sequence of a human G-protein coupled receptor
```

```
(HTADX50) and its diagnostic and therapeutic uses
     Bergsma, Derk J.; Ellis, Catherine E.
IN
PΑ
     Smithkline Beecham Corp., USA
SO
     Eur. Pat. Appl., 24 pp.
     CODEN: EPXXDW
DT
     Patent
LΑ
     English
ΙC
     ICM C12N015-12
          C07K014-705; A61K038-17; C12Q001-68; C12N015-11; C07K016-28;
          A61K048-00; G01N033-74
     3-3 (Biochemical Genetics)
     Section cross-reference(s): 6, 13, 63
FAN.CNT 1
     PATENT NO.
                     KIND DATE
                                          APPLICATION NO. DATE
     _____ ____
                            19980819
ΡI
     EP 859053
                      A1
                                          EP 1997-309253
                                                           19971118
         R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     US 5910430
                     Α
                           19990608
                                           US 1997-788750
                                                            19970124
     JP 10304887
                      A2
                            19981117
                                           JP 1998-51208
                                                            19980126
PRAI US 1997-788750
                      19970124
     HTADX50 polypeptides and polynucleotides and methods for producing such
     polypeptides by recombinant techniques are disclosed. The cDNA encoding
     human HTADX50 was first identified from a human activated T-cell cDNA
     library, and contains an open reading frame encoding a protein
     of 330 amino acids with a deduced mol. wt. of 37.1 kDa. HTADX50 has
about
     28% identity in 293 amino acid residues with the thrombin receptor, and
is
     also homologous to platelet-activating factor receptor and the
     ATP receptor; the cDNA has about 60.1% identity in 972 nucleotide
     with human B-cell receptor cDNA and is also homologous to interleukin-8
     receptor cDNA. Also disclosed are methods for utilizing HTADX50
     polypeptides and polynucleotides in the design of protocols for the
     treatment of infections such as bacterial, fungal, protozoan and viral
     infections, particularly infections caused by HIV-1 or HIV-2; pain;
     cancers; anorexia; bulimia; asthma; Parkinson's disease; acute heart
     failure; hypotension; hypertension; urinary retention; osteoporosis;
     angina pectoris; myocardial infarction; ulcers; asthma; allergies; benign
     prostatic hypertrophy; and psychotic and neurol. disorders, including
     anxiety, schizophrenia, manic depression, delirium, dementia,
     severe mental retardation and dyskinesias, such as Huntington's disease
or
     Gilles dela Tourett's syndrome, among others and diagnostic
     assays for such conditions.
st
     G protein coupled receptor HTADX50 human; sequence receptor
     HTADX50 cDNA human
IT
     G protein-coupled receptors
     RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic
     use); BIOL (Biological study); PREP (Preparation); USES (Uses)
        (HTADX50; cloning and cDNA sequence of a human G-protein
        coupled receptor (HTADX50) and its diagnostic and therapeutic
        uses)
ΙT
    Diagnosis
     Drug screening
     Drugs
     Molecular cloning
```

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(cloning and cDNA sequence of a human G-protein coupled
        receptor (HTADX50) and its diagnostic and therapeutic uses)
TΤ
     Antibodies
     Primers (nucleic acid)
     Probes (nucleic acid)
     RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical
     study); BIOL (Biological study); USES (Uses)
        (cloning and cDNA sequence of a human G-protein coupled
        receptor (HTADX50) and its diagnostic and therapeutic uses)
IΤ
     Mutation
        (detn. of mutations in diseases; cloning and cDNA sequence of a human
        G-protein coupled receptor (HTADX50) and its
      diagnostic and therapeutic uses)
IΤ
     cDNA sequences
        (for human G-protein coupled receptor HTADX50)
IT
     Protein sequences
        (of human G-protein coupled receptor HTADX50)
TΤ
     199397-48-3P
     RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic
     use); BIOL (Biological study); PREP (Preparation); USES (Uses)
        (amino acid sequence; cloning and cDNA sequence of a human G-
      protein coupled receptor (HTADX50) and its diagnostic
        and therapeutic uses)
IT
     211806-14-3P
     RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic
     use); BIOL (Biological study); PREP (Preparation); USES (Uses)
        (nucleotide sequence; cloning and cDNA sequence of a human G-
      protein coupled receptor (HTADX50) and its diagnostic
        and therapeutic uses)
     ANSWER 5 OF 5 MEDLINE
                                                         DUPLICATE 2
ΑN
     88204208
                  MEDLINE
DN
     88204208
     Sialic acid in platelets of schizophrenic patients.
     Sirota P; Bessler H; Allalouf D; Djaldetti M; Levinsky H
     Yehuda Abrabanel Mental Health Center, Bat Yam, Israel.
     PROGRESS IN NEURO-PSYCHOPHARMACOLOGY AND BIOLOGICAL PSYCHIATRY, (1988) 12
SO
     (1) 103-7.
     Journal code: Q45. ISSN: 0278-5846.
CY
     ENGLAND: United Kingdom
     Journal; Article; (JOURNAL ARTICLE)
DT
LΑ
     English
FS
     Priority Journals
EΜ
     198808
AB
     1. Nerve cell membrane sialic acid is involved in the activity of nervous
     tissue by its capacity to bind Ca ions and positively charged biogenic
     amines. 2. Blood platelets may serve as a model for
     amine-storing neurons. 3. The purpose of the present study was to
     determine the sialic acid content of platelets of schizophrenic
     patients in view of reports showing a reduced serotonin uptake by their
     platelets. 4. To this end platelets were isolated from
     the blood of 26 schizophrenic patients (13 males and 13 females) of
     various diagnostic subtypes and from 21 healthy subjects, and
     sialic acid was determined after hydrolysis at 80 degrees for 1 h in 0.1
    NHCl. 5. The results showed significantly lower contents of sialic acid
in
     the patients as compared to controls calculated both per 10(8) cells and
     per mg protein (18% and 25% lower, respectively) and appear to
```

be in line with the reduced serotonin uptake in their cells in schizophrenia. 6. There were no appreciable differences between sexes and between the various subtypes of this disease. CTCheck Tags: Female; Human; Male Adult *Blood Platelets: ME, metabolism *Schizophrenia: BL, blood Serotonin: BL, blood *Sialic Acids: BL, blood RN 131-48-6 (N-Acetylneuraminic Acid); 50-67-9 (Serotonin) CN 0 (Sialic Acids) => d his (FILE 'HOME' ENTERED AT 12:00:37 ON 17 APR 2001) FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 12:00:54 ON 17 APR 2001 L1 1290572 S DIAGNOSTIC L212154 S L1 AND SCHIZOPHRENIA L3 136 S L2 AND PLATELET 0 S L3 AND ISOLECTIC POINT L4L51 S L3 AND PI L6 85 DUP REMOVE L3 (51 DUPLICATES REMOVED) L7 8 S L3 AND PROTEIN 18 5 DUP REMOVE L7 (3 DUPLICATES REMOVED) => s 16 and DHT L9 0 L6 AND DHT => s 16 and delayed type hypersensitivity reaction L10 O L6 AND DELAYED TYPE HYPERSENSITIVITY REACTION => s 16 and DTH L111 L6 AND DTH => d 111L11 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS 1999:390463 CAPLUS DN 131:16115 Skin test for schizophrenia TIIN Shinitzky, Meir; Deckmann, Michael PΑ Yeda Research and Development Co. Ltd., Israel SO PCT Int. Appl., 21 pp. CODEN: PIXXD2 DTPatent LΑ English FAN.CNT 1 PATENT NO. KIND DATE APPLICATION NO. DATE _____ -----ΡI WO 9930163 19990617 WO 1998-IL592 19981207 A1

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG AU 1999-14457 EP 1998-958394 AU 9914457 A1 19990628 19981207 20000920 EP 1036333 A1 19981207 R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, SE, IE A 20001010 BR 1998-13402 BR 9813402 19981207 PRAI IL 1997-122490 19971207 WO 1998-I L592 19981207 RE.CNT 2 (1) Burbaea, G; Zk Nevropatol Psikhiatr im S S Korsakova 1986, V86(1), P193 (2) Yeda Research and Development Company Ltd; WO 9713152 A 1997 CAPLUS => ---Logging off of STN---Executing the logoff script... => LOG Y COST IN U.S. DOLLARS SINCE FILE TOTAL ENTRY SESSION FULL ESTIMATED COST 39.71 39.86 DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL ENTRY SESSION CA SUBSCRIBER PRICE -2.35-2.35

STN INTERNATIONAL LOGOFF AT 12:08:18 ON 17 APR 2001